

Interaction of HapX with the CCAAT-binding complex--a novel mechanism of gene regulation by iron.

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Abstract

Iron homeostasis requires subtle control systems, as iron is both essential and toxic. In *Aspergillus nidulans*, iron represses iron acquisition via the GATA factor SreA, and induces iron-dependent pathways at the transcriptional level, by a so far unknown mechanism. Here, we demonstrate that iron-dependent pathways (e.g., heme biosynthesis) are repressed during iron-depleted conditions by physical interaction of HapX with the CCAAT-binding core complex (CBC). Proteome analysis identified putative HapX targets. Mutual transcriptional control between hapX and sreA and synthetic lethality resulting from deletion of both regulatory genes indicate a tight interplay of these control systems. Expression of genes encoding CBC subunits was not influenced by iron availability, and their deletion was deleterious during iron-depleted and iron-replete conditions. Expression of hapX was repressed by iron and its deletion was deleterious during iron-depleted conditions only. These data indicate that the CBC has a general role and that HapX function is confined to iron-depleted conditions. Remarkably, CBC-mediated regulation has an inverse impact

on the expression of the same gene set in *A. nidulans*, compared with *Saccharomyces cerevisiae*.

Beteiligte Forschungseinheiten

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